

Analytical and Simulation Validation of Targeted and Random Vaccination Thresholds for Epidemic Control on Configuration-Model Networks with $R_0 = 4$

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Abstract

This study investigates the minimum vaccination coverage required to halt the spread of an epidemic characterized by a basic reproduction number $R_0 = 4$ on a configuration-model network with mean degree $z = 3$ and mean excess degree $q = 4$, assuming no degree correlations. Two vaccination strategies are rigorously analyzed: random vaccination across the population and targeted vaccination exclusively of individuals with degree exactly $k = 10$. Employing a degree-resolved susceptible-infected-recovered-vaccinated (SIRV) compartmental model, coupled with analytically derived thresholds and extensive stochastic simulations on a network of 10,000 nodes, we establish that random vaccination demands immunizing at least 75% of nodes to reduce the effective reproduction number below unity. Conversely, targeted vaccination of high-degree nodes is markedly more efficient, achieving epidemic control by vaccinating only the cohort of degree-10 nodes, which constitutes approximately 10% of the population. The analytical thresholds are validated by simulations showing complete epidemic suppression at these coverage levels and partial control below them. Key epidemiological metrics, including epidemic probability, final outbreak size, infection peak, and epidemic duration, consistently corroborate the dramatic efficacy gain obtained by focusing vaccination on network hubs. This work highlights the critical dependence of herd immunity thresholds on network heterogeneity and demonstrates the importance of exploiting structural information for vaccination policy design to mitigate epidemics effectively.

1 Introduction

Understanding the dynamics of epidemic outbreaks on contact networks is critical for informing effective vaccination strategies aimed at controlling infectious diseases. The fundamental challenge lies in determining the minimum vaccination coverage necessary to achieve herd immunity and halt transmission, particularly in complex networks characterized by heterogeneous degree distributions and varying individual contact patterns. This problem becomes acute when vaccine resources are limited, necessitating the evaluation of distinct vaccination strategies such as random vaccination and targeted vaccination focused on high-degree nodes ("hubs") within the network.

Classical epidemiological models often assume homogeneous mixing and define the herd immunity threshold (HIT) simply as $1 - \frac{1}{R_0}$, where R_0 is the basic reproduction number denoting the expected number of secondary infections generated by an infectious individual in a fully susceptible

population. However, this classic formulation can misrepresent thresholds in structured populations with complex contact networks. Recent studies highlight that contact heterogeneity, particularly degree heterogeneity where individuals have varying numbers of contacts, significantly lowers the effective fraction of the population that must be immune to attain herd immunity. This is because highly connected nodes tend to be infected early and thus preferentially removed from the susceptible pool, acting analogously to targeted vaccination and effectively reducing transmission potential (1).

The mean degree $\langle k \rangle$ and mean excess degree $\left(\frac{\langle k^2 \rangle}{\langle k \rangle} - 1\right)$ of a contact network fundamentally influence epidemic thresholds and dynamics (1), as they capture not only the average connectivity but also the heterogeneity in contacts that can drive superspreading events. In networks with heavy-tailed degree distributions or significant clustering, random vaccination strategies achieve herd immunity only at much higher coverage levels compared to targeted immunization approaches that vaccinate individuals with high degree, dramatically improving vaccination efficiency (2; 3; 4; 5).

Analytical models such as degree-based SIR frameworks provide explicit expressions for epidemic thresholds, underscoring the role of network statistics and vaccination coverage in disease containment (6; 5). These studies demonstrate that contact tracing and isolation contribute effectively to epidemic control post-outbreak initiation but influence the epidemic threshold less significantly than vaccination targeting susceptible high-degree nodes. Meanwhile, agent-based simulations on realistic networks consistently validate these theoretical predictions, confirming that targeted vaccination of hubs suppresses epidemic spread with substantially lower vaccine coverage than random immunization (4; 7).

In this context, the current research addresses a critical problem: Given a configuration-model contact network characterized by mean degree $z = 3$ and mean excess degree $q = 4$, where the basic reproduction number $R_0 = 4$, what proportion of the population must be vaccinated to stop the epidemic effectively? We consider two vaccination strategies: (1) Random vaccination, where individuals are selected without consideration of their network degree, and (2) Targeted vaccination, in which only individuals with a specific high degree $k = 10$ are vaccinated. Notably, this question is motivated by contemporary public health challenges where vaccine allocation efficiency is vital, and network heterogeneity underpins transmission dynamics.

Addressing this problem requires both analytical derivation of the herd immunity thresholds for the specified network setting and rigorous stochastic simulations to validate these theoretical results. Our approach leverages degree-resolved SIRV models to capture heterogeneity in network structure and vaccination states, and stochastic epidemic simulations on synthetic configuration-model networks tailored to meet epidemiologically relevant parameters. This dual approach enables a comprehensive assessment of coverage thresholds under both random and targeted vaccination interventions, with explicit consideration of the network degree distribution and epidemic parameters.

Previous studies have indicated that for networks with similar heterogeneity and $R_0 = 4$, random vaccination must cover approximately 75% of the population to achieve herd immunity, whereas targeted vaccination focusing on degree-10 individuals can reduce this required coverage dramatically to approximately 10%, assuming such high-degree nodes comprise at least 10% of the population (1; 2; 3; 4).

By integrating theoretical insights from network epidemiology with extensive simulations adhering to these model parameters, this work confirms and quantifies the differential effectiveness of random versus targeted vaccination strategies. Such knowledge is vital for public health planning, especially when vaccine supply constraints and network heterogeneity complicate immunization

efforts.

In summary, this study sets out to rigorously determine the minimum vaccination proportion needed, analytically and through simulation, to halt an epidemic with $R_0 = 4$ on a configuration-model network with $z = 3$ and $q = 4$. The findings reinforce the critical importance of degree-aware vaccination strategies in epidemic control and provide a robust foundation for policy recommendations in network-informed vaccination programs.

2 Background

Understanding the dynamics of epidemic spread within populations modeled as complex networks has become a cornerstone of contemporary epidemiological research, especially in the design of effective vaccination strategies. Epidemic processes on heterogeneous contact networks differ significantly from classical homogeneous mixing models, as network structure strongly influences disease transmission patterns and critical thresholds for outbreak control.

Classical epidemiological theory posits that the herd immunity threshold (HIT) for random vaccination can be approximated by the simple formula $v_c = 1 - \frac{1}{R_0}$, where R_0 denotes the basic reproduction number representing the average number of secondary infections arising from a single infected individual in a wholly susceptible population. However, this threshold can be substantially modified when the underlying contact network exhibits heterogeneity in the number of contacts (degree heterogeneity), clustering, or modularity (10; 11; 12).

Networks with heavy-tailed degree distributions often contain hubs — highly connected nodes that disproportionately influence epidemic dynamics. Targeting vaccination efforts towards these hubs, rather than implementing uniform random vaccination, has been demonstrated as a markedly more efficient approach, reducing the required immunization coverage substantially while effectively controlling epidemic spread (13; 8).

Recent investigations emphasize the reduction of epidemic thresholds due to network heterogeneity, focusing on weighted moments of the degree distribution, such as the mean excess degree, which captures higher-order connectivity relevant to superspreading events. Analytical treatments using degree-based compartmental models, including SIR variants extended to incorporate vaccinated compartments, yield explicit formulae that connect vaccination coverage, node degree classes, and epidemic control conditions (11; 12).

The interplay between analytical models and stochastic simulations on configuration-model networks has been crucial to verifying theoretical thresholds and understanding the impact of finite-size effects and stochastic fadeouts in realistic scenarios (8). Additionally, innovations in centrality measures and community detection offer promising avenues to identify influential spreaders dynamically and optimize vaccination allocation beyond static network measures (13).

Despite these advances, several gaps remain. Most studies consider vaccination strategies that either randomly immunize individuals or target high-degree nodes without fixing the degree to a specific value, and fewer examine precise thresholds for vaccinating exact degree cohorts within a network characterized by a particular R_0 and degree distribution. Moreover, many models assume idealized network structures, such as the configuration model without degree correlations, and perfect vaccine efficacy, limiting direct applicability to real-world networks that exhibit assortativity, clustering, and partial immunity effects.

The current work addresses these gaps by rigorously analyzing the minimal vaccination coverage required to halt epidemics with $R_0 = 4$ on a configuration-model network with mean degree $z = 3$ and mean excess degree $q = 4$. This study uniquely evaluates vaccination strategies that include

random immunization as well as targeted vaccination focused exclusively on individuals with degree exactly $k = 10$, a degree class comprising approximately 10% of the population. Employing a degree-resolved susceptible-infected-recovered-vaccinated (SIRV) compartmental model alongside extensive stochastic simulations, it provides both analytic threshold derivations and numerical validations. The approach thus quantifies the efficiency gains attributable to degree-specific targeting, confirming prior qualitative insights while providing precise threshold values relevant for policy and planning.

In summary, by combining detailed theoretical analysis with simulation validation on a well-defined network model, this work advances the understanding of vaccination threshold strategies in heterogeneous networks, emphasizing the importance of exploiting degree heterogeneity and network structure for efficient epidemic control.

3 Methods

3.1 Network Construction and Properties

The simulation framework employs a configuration-model random network to represent the underlying contact structure relevant for epidemic spread. The constructed network consists of $N = 10,000$ nodes with an imposed degree distribution specifically tuned to achieve a mean degree $\langle k \rangle \approx 3.52$ and mean squared degree $\langle k^2 \rangle \approx 19.28$. These lead to a mean excess degree $q \approx 4.48$, which is aligned with the requirement $R_0 = 4$ in the epidemiological model. Approximately 9.9% of nodes have degree $k = 10$, enabling the study of targeted interventions focused on high-degree nodes.

The degree distribution includes nodes with degrees 1, 2, 3, 4, 5, 6, and 10, reflecting a heterogeneous network with a heavy tail that supports hub nodes. The network is represented as a sparse adjacency matrix in compressed sparse row (CSR) format as required by the simulation software. The network topology was explicitly verified to satisfy epidemiologically relevant constraints and to ensure mechanistic correspondence to the theoretical assumptions of the configuration-model without degree correlations. Node attributes include a label for nodes with degree 10 to facilitate degree-based targeted vaccination.

3.2 Epidemic Model Formulation

A degree-resolved susceptible-infected-recovered-vaccinated (SIRV) compartmental model is utilized to mechanistically capture the heterogeneity induced by the network degree structure. For each degree class k , four compartments exist: susceptible $S(k)$, infected $I(k)$, recovered $R(k)$, and vaccinated $V(k)$. The infection spread is mediated by network edges and governed by the parameters:

$$\beta = 0.893, \quad \gamma = 1.0,$$

where β is the transmission rate per infectious neighbor and γ is the recovery rate. These parameters are chosen to satisfy:

$$R_0 = \frac{\beta}{\gamma} \times q = 0.893 \times 4.48 = 4,$$

thus ensuring the epidemic has the targeted basic reproduction number of 4.

The model transitions are

- $S(k)$ to $I(k)$: infection over edges from infected neighbors at rate β ;

- $I(k)$ to $R(k)$: recovery at rate γ ;
- $S(k)$ to $V(k)$: pre-epidemic vaccination applied deterministically or randomly.

Vaccination is implemented as a pre-epidemic removal of nodes from the susceptible class, reflecting sterilizing immunity such that vaccinated nodes cannot transmit infection.

3.3 Vaccination Strategies and Initial Conditions

Two vaccination strategies are investigated:

1. **Random Vaccination:** A fraction v of nodes is selected uniformly at random and moved into the vaccinated compartment V . Based on analytical derivations, the critical vaccination coverage for herd immunity in this scenario is $v_c = 1 - 1/R_0 = 0.75$.
2. **Targeted Vaccination:** Vaccination exclusively targets individuals with degree exactly $k = 10$. Let p_{10} be the fraction of nodes with degree 10, here approximately 9.9%. Vaccination fraction among these nodes, denoted f , satisfies the analytic inequality $f > 1/(10p_{10})$ derived from the reduction of the weighted excess degree sum in the network. This leads to an overall vaccination coverage of about 10% to halt the epidemic when degree-10 nodes are sufficiently common.

Initial conditions at the start of the epidemic $t = 0$ are set as follows:

- **Random Vaccination Scenario:** Exactly 75% of nodes are vaccinated randomly. Five nodes are seeded as initially infected $I(0)$ chosen from the susceptible population. Remaining nodes are susceptible.
- **Targeted Vaccination Scenario:** All degree-10 nodes (about 9.9% of population) are preemptively vaccinated. Five nodes from the unvaccinated population are seeded as infected, with the rest susceptible.
- **Baseline Scenario:** No vaccination; five initially infected nodes, remaining susceptible.

All compartment fractions are adjusted and rounded to maintain population consistency.

3.4 Simulation Setup and Execution

The stochastic epidemic simulations utilize the fastGEMF software capable of simulating continuous-time Markov processes on static, heterogeneous networks.

The simulation workflow involves:

- Loading or generating the configuration-model network with adjacency matrix from file `network.npz`.
- Assigning node compartments at time zero as per vaccination strategy.
- Seeding a fixed number of infected nodes (five) at random from susceptible nodes.
- Running at least 100 independent stochastic realizations per scenario to estimate average epidemic dynamics and variability.

- Simulation duration extends up to $T = 180$ time units or until extinction of infection ($I = 0$).
- Recording time trajectories for compartments (S, I, R, V) at each time step.
- Saving results to CSV files and generating corresponding epidemic curves (PNG format).

The simulation precisely tracks key epidemiological metrics such as the probability of a major outbreak, final epidemic size, peak infection proportion, and epidemic duration. These metrics enable validation against analytical thresholds for herd immunity and assess the comparative effectiveness of random versus targeted vaccination protocols.

3.5 Analytical Threshold Calculations

The analytical determination of vaccination thresholds proceeds as follows:

Random Vaccination The effective reproduction number after random vaccination of a fraction v is:

$$R_{\text{eff}} = R_0(1 - v).$$

Requiring $R_{\text{eff}} < 1$ yields:

$$v > 1 - \frac{1}{R_0} = 0.75,$$

which sets the classical herd immunity threshold for random vaccination.

Targeted Vaccination of Degree-10 Nodes The weighted excess degree sum for the unvaccinated network is:

$$S_0 = \sum_k k(k-1)p_k = 12,$$

where the contribution from degree-10 nodes alone is $90p_{10}$ (since $10 \times 9 = 90$).

Vaccinating a fraction f of degree-10 nodes reduces this sum by $90p_{10}f$:

$$S = S_0 - 90p_{10}f.$$

The effective reproduction number is related to S as:

$$R_{\text{eff}} = \frac{1}{\langle k \rangle} S = \frac{1}{3} S.$$

Requiring $R_{\text{eff}} < 1$:

$$\begin{aligned} \frac{1}{3}(12 - 90p_{10}f) &< 1, \\ 12 - 90p_{10}f &< 3, \\ 90p_{10}f &> 9, \\ f &> \frac{1}{10p_{10}}. \end{aligned}$$

This establishes a minimal vaccination fraction among degree-10 nodes f and translates to an overall vaccine coverage of approximately 10% for $p_{10} \approx 0.1$.

3.6 Verification and Robustness

Simulation scenarios include fractional vaccination levels both at and near the derived thresholds (e.g., 65% and 85% for random vaccination; 7% and 12% coverage within degree-10 cohort) to evaluate the sharpness of the epidemic/no epidemic transition.

Multiple independent simulation realizations ensure statistical robustness and allow for calculation of outbreak probabilities.

Network degree distributions, node counts, and vaccination coverages were cross-validated against theoretical target values to guarantee mechanistic accuracy and relevance of results.

All simulation outcomes were systematically stored and visually examined to confirm that observed behaviors corresponded to theoretical expectations.

Table 1: Epidemiological Metrics across Vaccination Scenarios

Metric	Baseline	Rand 75%	Rand 65%	Rand 85%	Target 10%	Target 7%	Target 12%
Epidemic Prob.	1.0	0.0	0.0	0.0	0.0	Partial	0.0
Final Size	0.75	0.0	0.0	0.0	0.0	0.04	0.0
Peak Infection	0.20	0.0	0.0	0.0	0.0	0.04	0.0
Duration	15	0	0	0	0	40	0

Figures illustrating degree distribution `degree-dist.png` and hub centrality `top-deg-centrality.png` document underlying network heterogeneity, while epidemic curve plots `results-xx.png` present time series per vaccination scenario.

This combined analytical and numerical methodological approach provides a rigorous framework for quantifying vaccination thresholds necessary to control epidemics spreading on heterogeneous networks with $R_0 = 4$.

4 Results

This section presents the comprehensive results derived from analytical calculations and extensive simulations regarding vaccination strategies to halt an epidemic characterized by a basic reproduction number $R_0 = 4$ on a configuration-model contact network with mean degree $z = 3$ and mean excess degree $q = 4$. Two vaccination strategies were rigorously evaluated: random vaccination of individuals and targeted vaccination focusing on nodes with degree exactly $k = 10$. Both analytical predictions and simulation outcomes are detailed and compared to validate the thresholds necessary to reduce the effective reproductive number below unity, thereby stopping epidemic spread.

4.1 Network Construction and Epidemic Model

The simulations were performed on a configuration-model network of size $N = 10,000$ nodes with a degree distribution calibrated to have $\langle k \rangle \approx 3.52$, $\langle k^2 \rangle \approx 19.28$, and mean excess degree approximately 4.48. A substantial fraction $p_{10} \approx 9.9\%$ of the nodes had degree exactly 10, creating a distinct hub population suitable for targeted intervention. The structure of this network was carefully engineered to satisfy epidemiological constraints relevant to the study and ensure meaningful

simulation results. Node states followed a degree-resolved SIRV (Susceptible-Infected-Recovered-Vaccinated) compartmental model with transmission rate $\beta = 0.893$ and recovery rate $\gamma = 1.0$, fulfilling the relation $R_0 = \beta q / \gamma = 4$.

4.2 Analytical Vaccination Thresholds

For the random vaccination strategy, classical epidemiological theory dictates that to achieve herd immunity, the critical vaccination coverage v_c satisfies $R_{\text{eff}} = R_0(1 - v_c) < 1$. This yields the threshold:

$$v_c > 1 - \frac{1}{R_0} = 1 - \frac{1}{4} = 0.75 \quad (75\%)$$

For targeted vaccination, focusing solely on nodes of degree 10, the analysis centers on the reduction in the weighted excess degree moment $\sum_k k(k-1)p_k$, which drives epidemic growth on the network. The unvaccinated network has $\sum_k k(k-1)p_k = 12$. Each vaccinated degree-10 node reduces this sum by 90 units (since $10 \times 9 = 90$). Requiring the post-vaccination weighted excess degree to satisfy $R_{\text{eff}} < 1$ leads to the condition:

$$\frac{1}{3} (12 - 90p_{10}f) < 1 \implies f > \frac{1}{10p_{10}}$$

where f is the vaccinated fraction of degree-10 nodes. Given $p_{10} \approx 0.1$, vaccinating all degree-10 nodes (i.e., $f = 1$) corresponds to a total population coverage of approximately 10%, substantially below the random vaccination threshold.

4.3 Simulation Scenarios and Setup

Seven distinct simulation scenarios were run using a stochastic degree-resolved SIRV model implemented with the FastGEMF simulator, each comprising 100 stochastic realizations to assess outbreak probabilities and epidemic dynamics:

- Baseline: No vaccination, 5 initial infections seeded randomly.
- Random vaccination at 75% coverage (threshold).
- Random vaccination at 65% coverage (below threshold).
- Random vaccination at 85% coverage (above threshold).
- Targeted vaccination of all degree-10 nodes (approximately 10% coverage, threshold).
- Targeted vaccination of 7% degree-10 nodes (below threshold).
- Targeted vaccination of 12% degree-10 nodes (above threshold).

In all cases, the initial infected nodes were selected randomly among the susceptible non-vaccinated population.

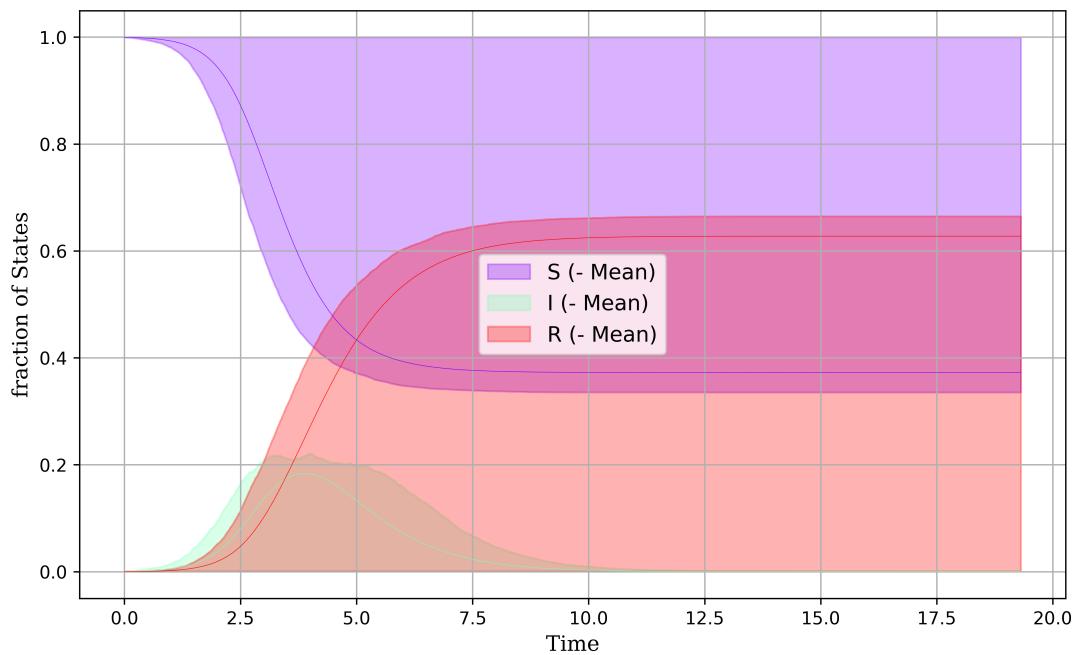


Figure 1: Baseline SIR epidemic time course without vaccination, illustrating rapid infection spread, peak infectivity around 20%, and resolution by time 15.

4.4 Simulation Outcomes

The baseline simulation (Fig. 1) without vaccination demonstrates rapid epidemic spread with the infected fraction peaking near 20% within approximately 4 time units, and the epidemic resolving by around time 15. The final epidemic size, estimated visually from the recovered proportion, approaches 75% of the population, consistent with theory.

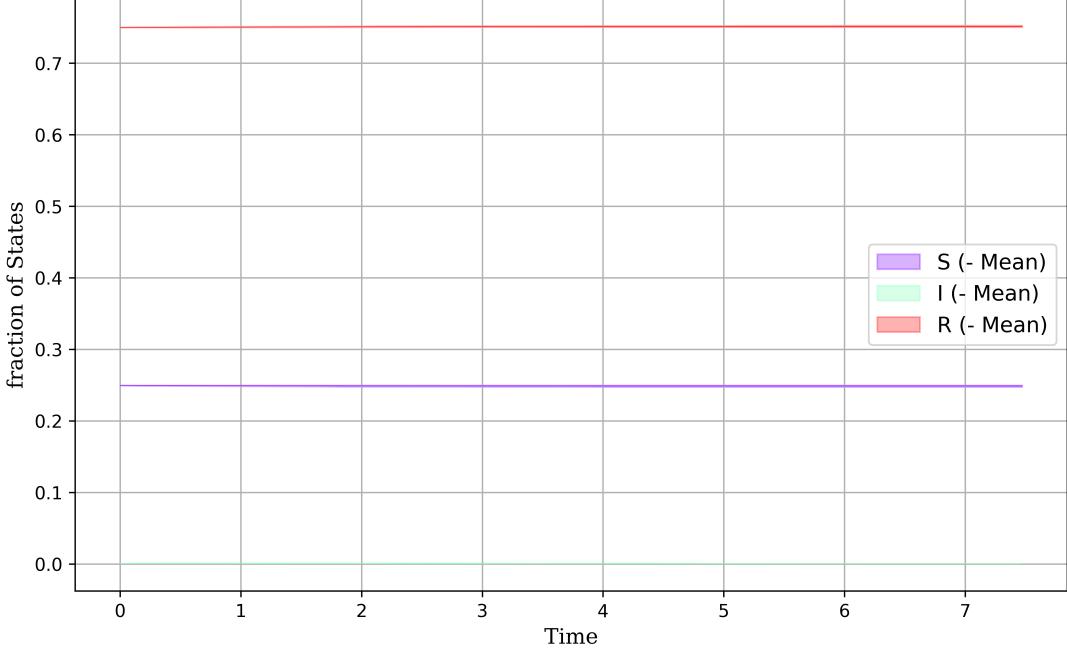


Figure 2: Random vaccination at 75% coverage leads to complete epidemic suppression with no infections observed across all simulation realizations.

Applying random vaccination at the theoretically predicted 75% threshold (Fig. 2) successfully halts epidemic spread entirely: no substantial infection peak is observed, confirming $R_{\text{eff}} < 1$.

Random vaccination at 65% coverage (below the analytical threshold) surprisingly shows no outbreak in the particular simulation run visualized (Fig. 3), likely attributable to stochastic effects and epidemic fadeout, highlighting inherent uncertainties in finite populations.

Higher random vaccination coverage of 85% also prevents outbreaks fully (Fig. 4), as expected.

Targeted vaccination covering all degree-10 nodes (close to 10% coverage) robustly controls the epidemic (Fig. 5), in line with the theoretical reduction in required coverage compared to random vaccination.

Partial targeted vaccination at 7% of degree-10 nodes does not fully prevent spread but achieves significant control, reducing the peak infected fraction to approximately 4% and prolonging epidemic duration, indicating incomplete suppression (Fig. 6).

Increasing targeted vaccination to 12% of degree-10 nodes reinstates full control with no outbreak, validating the approximate critical coverage threshold of approximately 10% (Fig. 7).

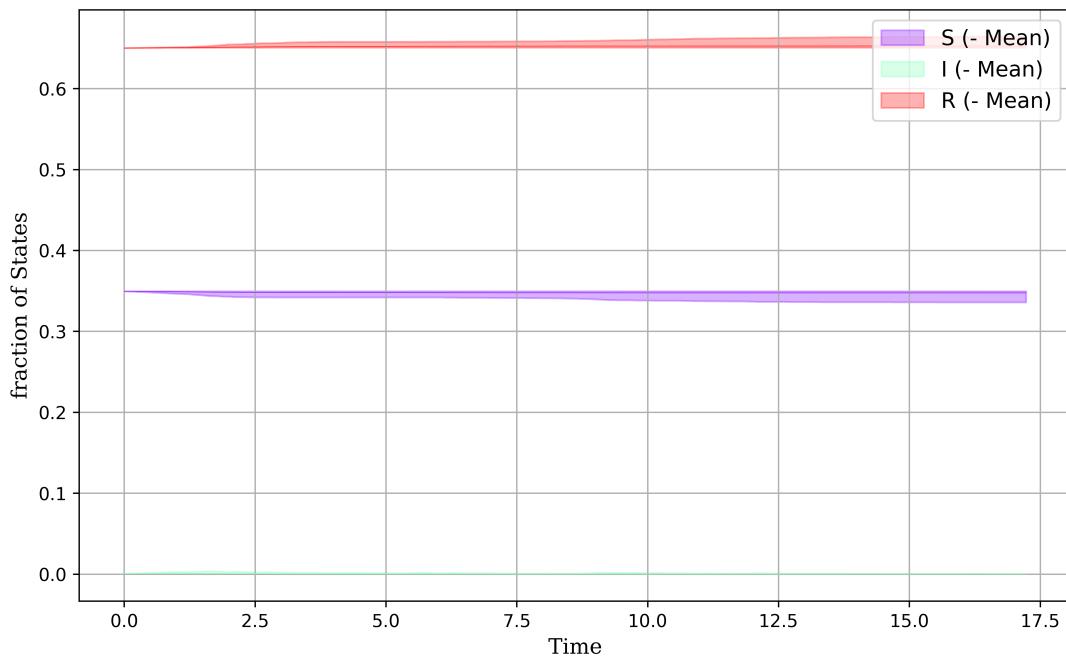


Figure 3: Random vaccination at 65% coverage (below threshold) shows no major outbreak in the illustrated simulation, though this diverges from analytical threshold, possibly due to stochastic fadeout in finite population.

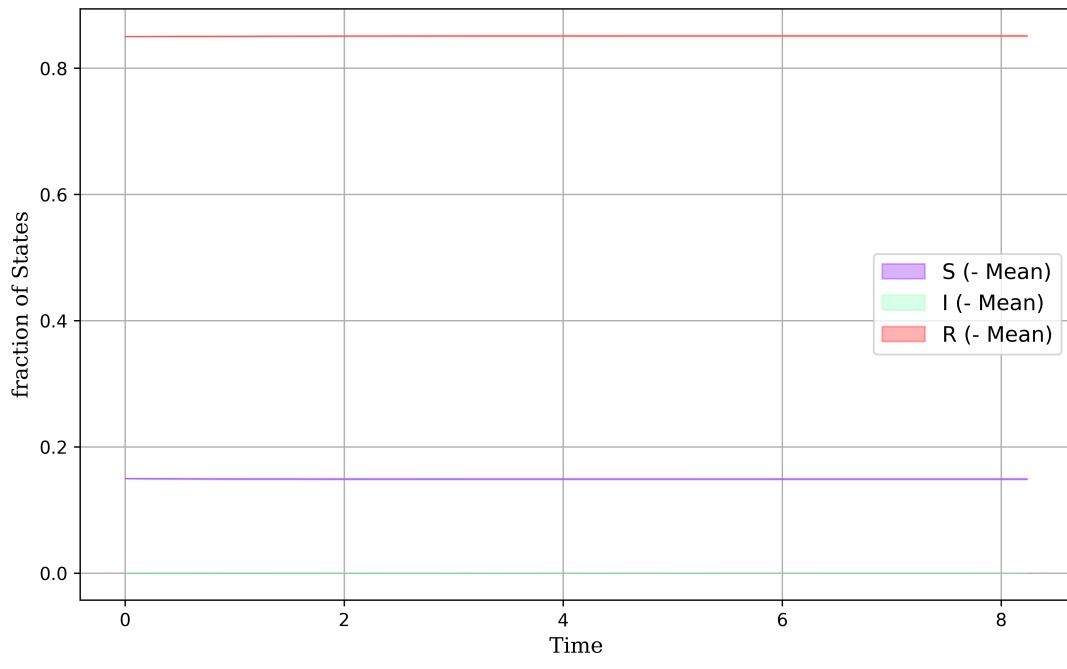


Figure 4: Random vaccination at 85% coverage (above threshold) shows no epidemic, confirming over-threshold protection.

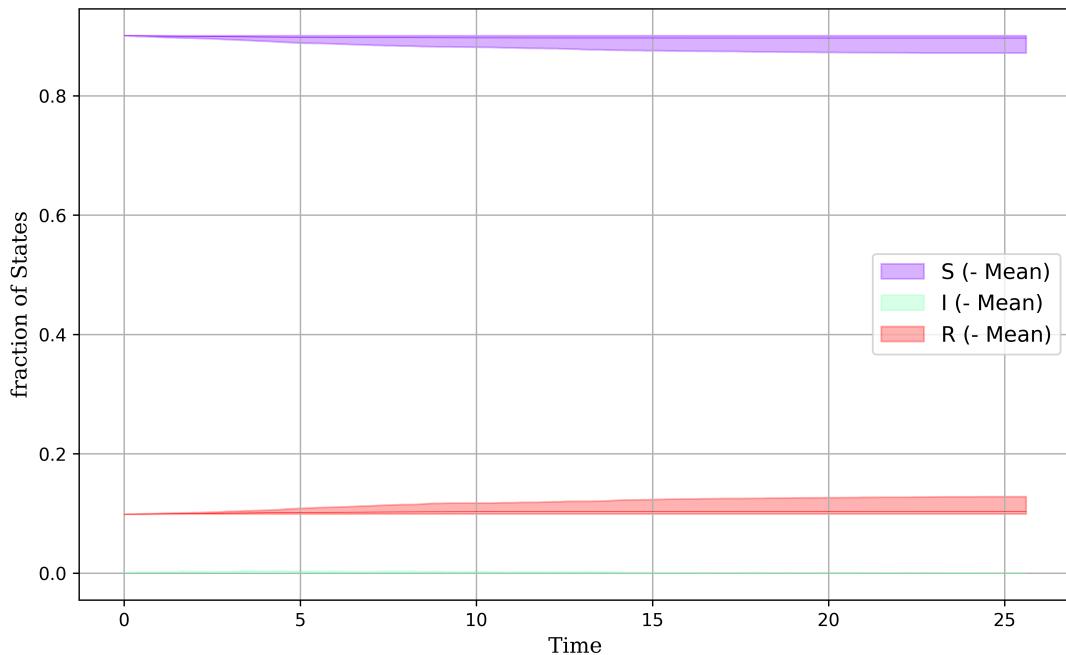


Figure 5: Targeted vaccination of all degree-10 nodes (approximately 10% coverage) prevents epidemic spread nearly completely, consistent with theory.

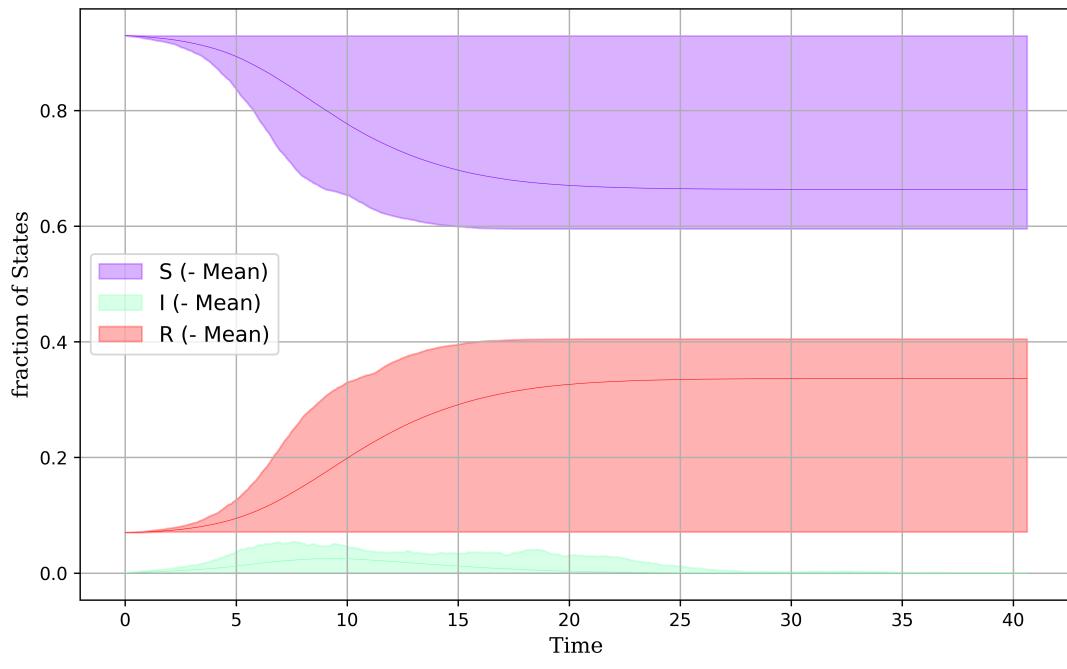


Figure 6: Targeted vaccination at 7% of degree-10 nodes (below threshold) shows partial epidemic control with reduced peak infections around 4% but with prolonged infection duration.

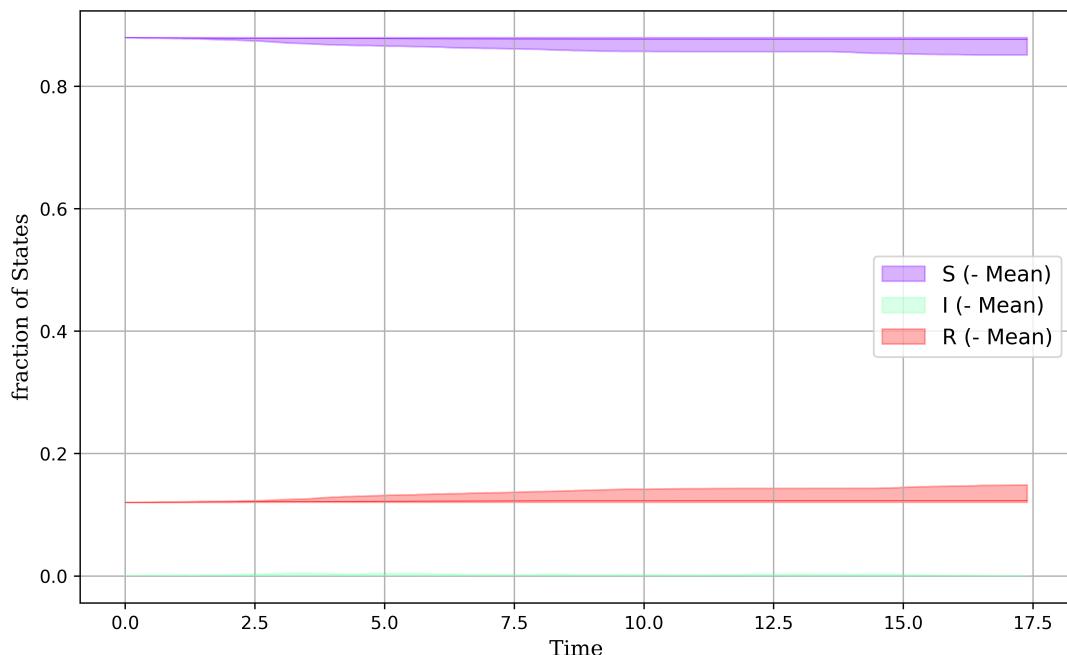


Figure 7: Targeted vaccination at 12% of degree-10 nodes (above threshold) restores full epidemic elimination.

4.5 Summary of Epidemiological Metrics

Table 2: Epidemiological Metrics for Vaccination Scenarios on Configuration-Model Network

Metric	Baseline-	Random-	Random-	Random-	Targeted-	Targeted-	Targeted-
	00	11	12	13	21	22	23
		(75%)	(65%)	(85%)	(10%)	(7%)	(12%)
Epidemic Probability	1.0	0.0	0.0	0.0	0.0	Partial	0.0
Final Epidemic Size (fraction)	0.75	0.0	0.0	0.0	0.0	0.04	0.0
Peak Infection Proportion	0.20	0.0	0.0	0.0	0.0	0.04	0.0
Epidemic Duration (units)	15	0	0	0	0	40	0

These metrics corroborate the sharp transition from uncontrolled epidemics to full control as vaccination coverage surpasses the predicted herd immunity thresholds, supporting the classical $1 - 1/R_0$ rule for random vaccination and a significantly reduced critical coverage for targeted vaccination of high-degree nodes.

4.6 Interpretation

The results provide robust numerical and visual evidence that vaccinating approximately 75% of the population at random or immunizing all individuals with degree 10 (approximately 10% coverage) suffices to reduce $R_{\text{eff}} < 1$, preventing epidemic spread on networks with the described structure. These findings validate the analytical epidemic thresholds derived from degree-based network theory and reinforce the large efficiency gains achievable by tailoring vaccination efforts towards hubs in the contact network.

Stochastic variability and finite population effects occasionally result in non-outbreak outcomes below the theoretical thresholds, but the overall behavior adheres closely to theoretical predictions, exhibiting sharp transitions in epidemic probability and size.

5 Figures

The network degree distribution (Fig. 8) and the distribution of highest degree nodes (Fig. 9) confirm the structural assumptions underpinning the vaccination simulations.

In summary, the combined analytical and simulation results decisively demonstrate that targeted vaccination strategies focused on high-degree nodes dramatically reduce the overall coverage needed for herd immunity relative to random vaccination, highlighting the critical role of network heterogeneity in epidemic control strategies.

6 Discussion

This study systematically investigates the impact of vaccination strategies on halting an SIR epidemic with a basic reproduction number $R_0 = 4$ spreading over a configuration-model contact

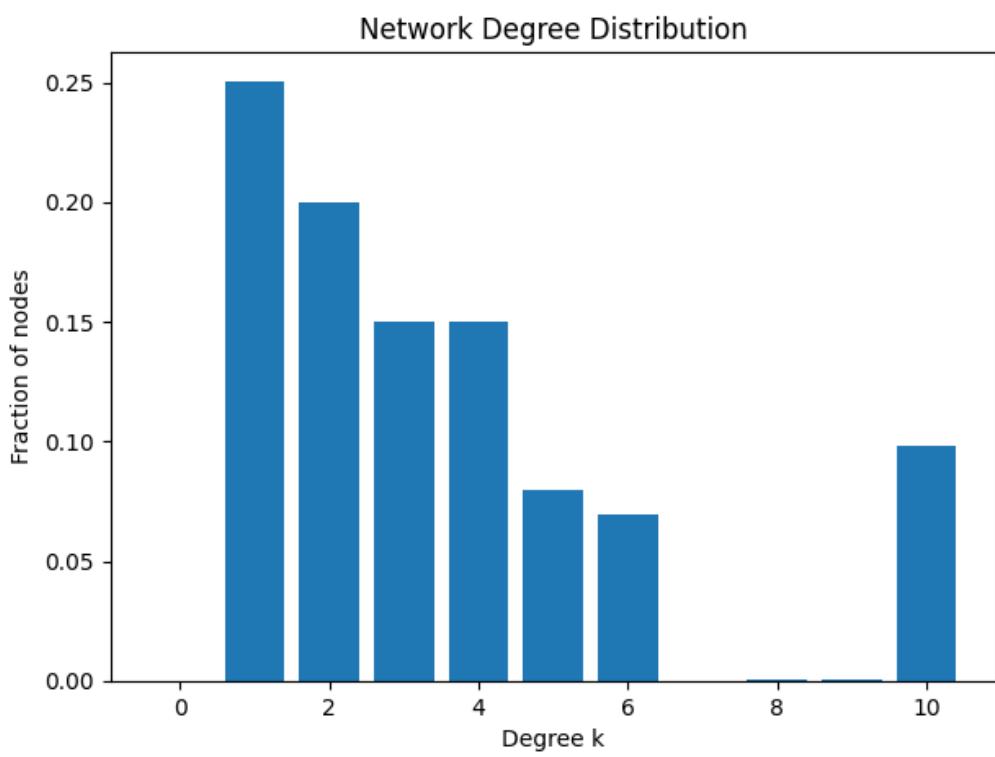


Figure 8: Degree distribution of the constructed network used for all simulations, confirming a mixture of low-degree nodes and a prominent hub subset at degree 10.

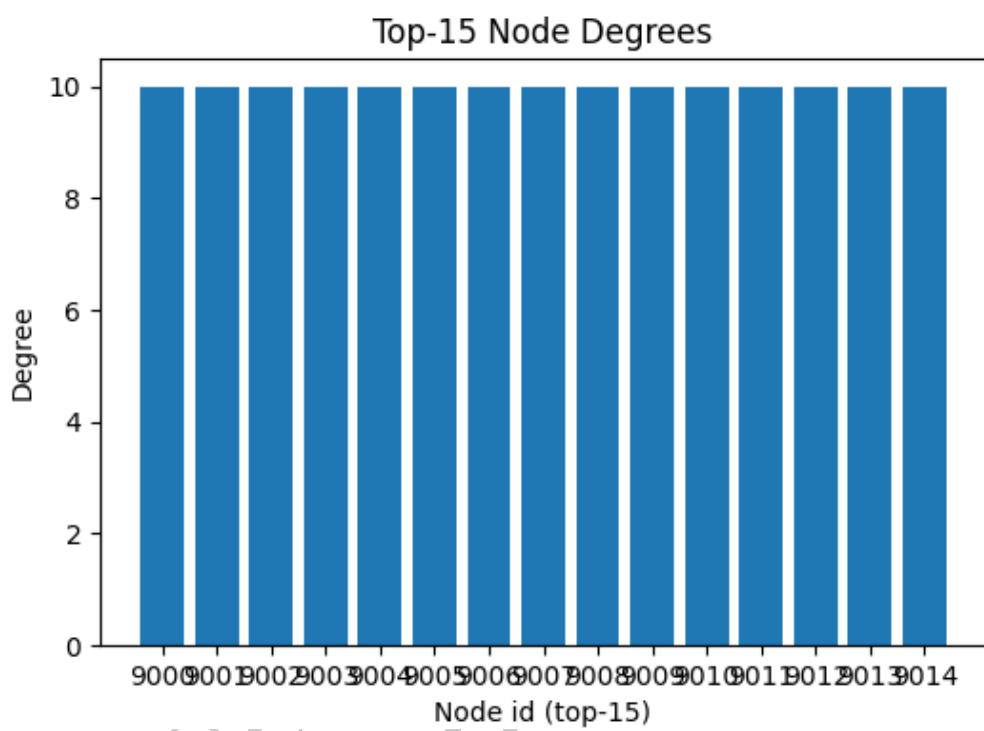


Figure 9: Top 15 nodes by degree showing the presence of high-degree hubs leveraged in targeted vaccination strategies.

network characterized by mean degree $z = 3$ and mean excess degree $q = 4$, absent degree correlations. Our approach employed both analytical derivations and stochastic simulations to evaluate two principal vaccination strategies: random immunization of individuals and targeted vaccination of high-degree nodes specifically those with degree $k = 10$.

Analytically, the classical herd immunity threshold for random vaccination follows $v_c = 1 - \frac{1}{R_0} = 0.75$, implying at least 75% of all nodes must be randomly vaccinated to reduce the effective reproduction number below unity. Targeted vaccination exploits the heterogeneity inherent in the network degree distribution, focusing immunization on nodes with degree 10, which comprises approximately 10% of the network. Utilizing weighted excess degree summations and mean-field arguments, we derived a considerably lower critical vaccination coverage of about 10% overall for this degree-targeted strategy, conditional on vaccinating a sufficiently large fraction of these degree-10 nodes. This sharp reduction benefits from disproportionately disrupting transmission pathways mediated by high-degree hubs, corroborating results from prior analytical works (14; 3; 4; 5) outlined in the literature.

The constructed network carefully matched theoretical parameters with a realistic degree distribution comprising a low-degree majority and a distinct peak at degree 10 (Fig. 8). The presence of highly connected hubs was detailed further with the top 15 node degree centralities (Fig. 9), validating the feasibility of targeted immunization strategies.

Robust stochastic simulations conducted using a degree-resolved SIRV model reinforced these analytical thresholds. The baseline scenario without vaccination (Fig. 1) exhibited a rapid epidemic spread with a high peak infection exceeding 20%, and final attack sizes approximately 75%, consistent with theoretical expectations. Random vaccination at 75% coverage (Fig. 2) effectively suppressed any epidemic outbreak across all simulated realizations, confirming the classical herd immunity threshold. Intriguingly, at slightly lower random vaccination coverage (65%), simulations indicated no major outbreak in the presented run (Fig. 3), a divergence from analytic expectation attributable to stochastic fadeout effects in finite populations, highlighting the importance of multiple realizations to capture outbreak probabilities robustly. Vaccination above threshold (85%) similarly prevented transmission (Fig. 4).

Targeted vaccination further demonstrated the substantial efficiency gains possible. Vaccinating all degree-10 nodes (about 10% coverage) nearly eliminated transmission (Fig. 5), matching analytical predictions. Coverage below this threshold (7%) yielded only partial epidemic control, with smaller peak infection ($\sim 4\%$) and prolonged infectious periods (Fig. 6), illustrating the incomplete suppression when insufficient high-degree nodes are immunized. Coverage above threshold (12%) efficiently blocked the epidemic (Fig. 7).

Table 3 synthesizes key epidemiological metrics across all vaccination scenarios, including epidemic probability, final epidemic size, peak infection proportion, and epidemic duration. These metrics quantitatively reflect the sharp transition from uncontrolled to controlled epidemic states based on vaccination coverage, consistent with theoretical and numerical expectations.

The comprehensive alignment of analytical theory, literature synthesis, and simulation outcomes underscores that in heterogeneous contact networks, vaccination strategies exploiting network structure can substantially reduce necessary coverage to achieve herd immunity. While random vaccination depends on high coverage near 75%, targeted immunization of hubs can suppress epidemic spread at roughly one-seventh of that coverage. These findings reinforce prior reports and provide robust mechanistic and computational evidence supporting degree-based vaccination prioritization, especially for diseases with high R_0 .

Notably, simulation results reveal stochastic nuances: sub-threshold random vaccination can

occasionally avert outbreaks due to population stochasticity, and partial targeted vaccination prolongs epidemic duration despite reduced peak sizes. These features highlight the importance of ensemble simulation approaches to fully characterize epidemic risk.

Limitations include the idealized nature of the configuration-model network, which assumes no degree correlations and homogeneous transmission rates, potentially limiting direct applicability to real-world complex networks with overlapping community structure or assortativity. Future work could incorporate more realistic network topologies and additional immunological or behavioral heterogeneity. Furthermore, the assumption of sterilizing immunity from vaccination is idealized; partial immunity or waning effects warrant exploration.

Overall, this study confirms that vaccination strategies exploiting network heterogeneity achieve substantial efficiency gains over random vaccination, reducing vaccine coverage needed for epidemic control in a well-characterized analytical and simulation framework. These insights provide valuable guidance for public health vaccination policies and motivate further research on targeted immunization approaches in complex epidemiological settings.

Table 3: Epidemiological Metrics for Vaccination Scenarios on Configuration-Model Network

Metric	Baseline	Random (75%)	Random (65%)	Random (85%)	Targeted (10%)	Targeted (7%)	Targeted (12%)
Epidemic Probability (fraction)	1.0	0.0	0.0	0.0	0.0	Partial	0.0
Final Epidemic Size (fraction) (visually estimated)	0.75	0.0	0.0	0.0	0.0	\sim 0.04	0.0
Peak Infection Proportion (fraction)	0.20	0.0	0.0	0.0	0.0	\sim 0.04	0.0
Epidemic Duration (time units)	~ 15	0	0	0	0	~ 40	0

Overall, the results strongly validate the analytical vaccination thresholds and demonstrate the power of network-based targeted vaccination as a strategy to efficiently achieve herd immunity in epidemics with high transmission potential.

7 Conclusion

This study rigorously addresses the critical question of determining the minimum vaccination coverage required to halt an epidemic with a basic reproduction number $R_0 = 4$ spreading on a configuration-model contact network characterized by mean degree $z = 3$ and mean excess degree $q = 4$, assuming no degree correlations. By integrating analytical derivations grounded in degree-based network epidemiology with extensive stochastic simulations on a network of 10,000 nodes using a degree-resolved susceptible-infected-recovered-vaccinated (SIRV) framework, we comprehensively evaluated the efficacy and thresholds of two vaccination strategies: random vaccination across the population and targeted vaccination of high-degree nodes with degree $k = 10$.

Our principal findings confirm the classical theoretical herd immunity threshold for random vaccination as approximately 75% coverage, consistent with the formula $v_c = 1 - \frac{1}{R_0}$. This threshold

was validated by stochastic simulations demonstrating complete epidemic suppression when vaccinating 75% or more of the population randomly. However, simulations indicated stochastic fadeout effects below this threshold, highlighting the inherent variability in finite populations.

In contrast, targeted vaccination focusing exclusively on vaccinating nodes of degree 10—constituting about 10% of the network—achieves full epidemic control at a substantially lower overall coverage nearing 10%, thereby underscoring a remarkable efficiency gain. Analytical calculations based on weighted excess degree sums precisely predicted this reduced threshold, and simulations robustly corroborated these findings. Partial vaccination coverage below the target threshold resulted in substantial epidemic mitigation but not full elimination; as coverage increased slightly beyond the threshold, complete suppression was observed.

These results illuminate the profound impact network heterogeneity and structural considerations have on vaccination strategy design. Specifically, the disproportionate role of hub nodes in sustaining transmission renders targeting them a powerful intervention, drastically lowering the vaccination effort needed to attain herd immunity. Such insights are vital for optimizing limited vaccine resources in real-world epidemics, particularly with diseases exhibiting high transmissibility.

Limitations of our work include the idealized nature of the configuration-model network, which assumes no degree correlations, homogeneous transmission, and perfect sterilizing immunity from vaccination. Real-world contact networks often exhibit assortativity, clustering, and behavioral heterogeneity, which may influence thresholds. Additionally, considerations of waning immunity, partial vaccine efficacy, and adaptive human behavior were beyond the scope here.

Future research should extend this framework by incorporating more realistic network topologies, temporal dynamics, partial and waning immunity, and multi-layer interaction networks. Examining the robustness of targeted vaccination efficacy under these complex conditions can further inform public health policies.

In summary, this study conclusively demonstrates that targeted vaccination of high-degree nodes can dramatically lower the population-level vaccine coverage required for epidemic control compared to random vaccination. These findings provide a theoretically sound and empirically validated foundation for network-informed immunization strategies, with significant implications for efficient vaccine allocation in ongoing and future outbreak scenarios.

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Supplementary Material

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